

# Type 1 Brugada pattern electrocardiogram induced by hypokalemia

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### ABSTRACT

Coved-type ST-segment elevation in the right precordial leads are the characteristics of Brugada syndrome, an inherited arrhythmogenic ion channel disease, which could lead to ventricular arrhythmia and sudden death. Hypokalemia alone may induce Type 1 Brugada pattern electrocardiogram (EKG), and the association has rarely been reported. We describe a patient with hypokalemia 2.9 mmol/L and the appearance of new right bundle branch block pattern with coved ST-segment elevations with inverted T wave in leads V1-V2. Serum potassium was corrected and repeated EKG 6 h later revealed disappearance of Type 1 Brugada pattern. Although there is no definite value of serum potassium level that can induce Brugada pattern EKG, hypokalemia may unmask Type 1 Brugada EKG pattern. Awareness of its appearance should be made by all physicians since patients with a family history of arrhythmia or sudden cardiac death (SCD) are at the high risk of developing SCD.

**Keywords:** Brugada syndrome, hypokalemia, sudden cardiac death

### Introduction

Brugada syndrome (BS) is a fatal inherited arrhythmogenic disease due to syncope, ventricular arrhythmia, and sudden death. It is characterized by two electrocardiogram (EKG) patterns in leads V1-V3. Type 1 is a coved pattern and Type 2 is the saddleback pattern. The Type 1 pattern which is considered as a BS has a coved ST-segment elevation  $\geq 2$  mm followed by a down-sloping concave or rectilinear ST-segment with a negative T wave. The Type 2 pattern is characterized by a high take-off ( $r'$ ) that is  $\geq 2$  mm from the isoelectric baseline, followed by ST-segment elevation that is convex to the isoelectric baseline with elevation  $\geq 0.05$  mV, with variable T wave in lead V1 and positive or flat T wave in lead V2.<sup>[1]</sup>

### Case Report

A 39-year-old male with a medical history of hypertension and diabetes mellitus presented to the emergency department with

complaints of intermittent chest pain and dyspnea on exertion for 3 days. Chest pain was located at the left side of the chest, six out of ten in intensity, sharp in character without radiation. It was aggravated by breathing and coughing and relieved by nothing. Each episode of chest pain lasted for 30–60 min. The patient denied syncope, orthopnea, paroxysmal nocturnal dyspnea, palpitations, dizziness, headache, syncope, presyncope, history of heart attack, arrhythmia, seizure, blurred vision, urinary, or bowel problems. His home medications include ibuprofen as needed for chest pain and hydralazine 25 mg per os twice daily. He has an allergy to amlodipine and enalapril causing skin rash. Family history revealed his mother had an unknown cardiac problem, and his sister had sudden cardiac death (SCD) while she was at age 30.

Initial vital signs included temperature 98.7°F, pulse rate 96 beats per minutes, respiratory rate 22 breaths per minutes, blood pressure 130/90 mmHg, and oxygen saturation 92% on room air. He was put on nonrebreather mask with 40% of oxygen for hypoxia and oxygen saturation was maintained with 95%.

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Physical examination showed fast breathing with crepitation at basal lungs. Cardiovascular examination revealed normal apex beat without any thrill or heave. The point of maximal impulse was not displaced. On auscultation, normal regular first and second heart sound without murmur, rub, or gallop was noted. Head, neck, abdominal, and neurological examinations were within normal limits.

Laboratory tests showed normal complete blood count including normal white blood cells with differentials. Comprehensive metabolic panel revealed potassium 2.9 mmol/L, magnesium 2.7 mg/dL, sodium 136 mmol/L, chloride 101 mmol/L, carbon dioxide 28 mmol/L, blood urea nitrogen 10 mg/dL, creatinine 1 mg/dL, glomerular filtration rate 90 ml/min, calcium 9 mg/dL, phosphorus 3.1 mg/dL, 3 sets of troponin 6 h apart were 0.01 ng/mL, and B type natriuretic peptide 5 pg/mL. Coagulation profile and arterial blood gasses with 40% of oxygen with nonrebreather mask were within normal limit. Computed tomography of the chest revealed moderate to marked ground glass opacities in both lung fields and normal heart size. An echocardiogram was within normal limit. He was admitted due to hypoxemic respiratory failure secondary to bilateral interstitial pneumonia. An EKG revealed a new right bundle branch block pattern with coved ST-segment elevations and inverted T wave in leads V1-V2 consistent with Type 1 Brugada pattern EKG [Figure 1].

Hypokalemia was corrected with potassium chloride 40 mEq per os once and 20 mEq twice intravenously. Rechecked potassium was 3.6 mmol/L. Six hours later, EKG was repeated and Brugada pattern EKG was disappeared, and EKG returned to its baseline [Figure 2].

The patient was treated with antibiotics and antivirals for bilateral interstitial pneumonia likely due to viral or bacterial etiology. No other arrhythmia or reappearance of Brugada EKG pattern was found throughout hospital course. Potassium was found to be within normal limit for rest of hospital stay. No fever was noted throughout hospital course. Chest pain on admission day was thought to be pleuritic chest pain and patient was discharged. Although the patient has a strong family history of cardiac problem, patient and family members were recommended to

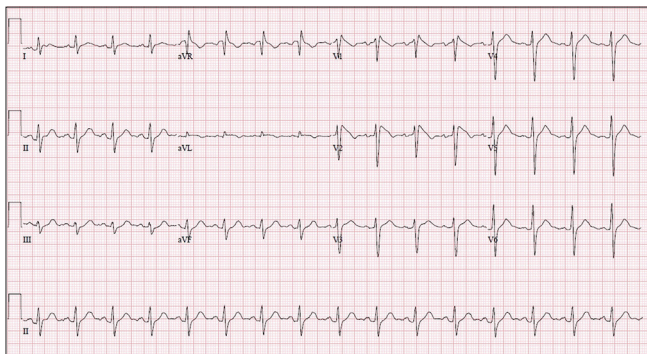
follow-up with an electrophysiologist for drug challenge test for possible BS, but they refused. Up to 5 months after discharge, the patient was contacted over the telephone and he was in good health without any syncope, chest pain, arrhythmia, or cardiac symptoms.

## Discussion

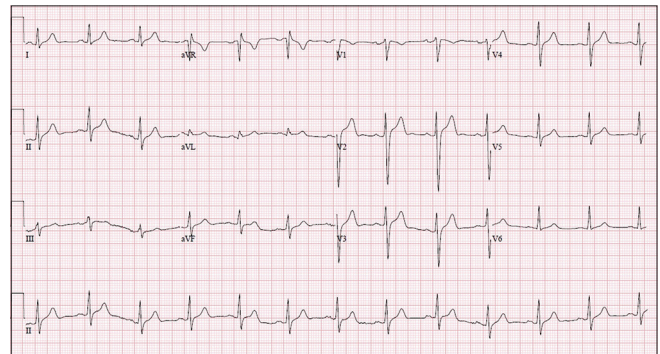
Type 1 Brugada pattern EKG is a rare condition, and the prevalence of the typical EKG changes of the Brugada pattern in two groups of urban populations in the United States was about 0.4 and 0.012%, respectively.<sup>[2]</sup> BS shows autosomal dominant inheritance with variable expression and found to have mutations in the SCN genes SCN5A and SCN10A which are encoding subunits of a cardiac sodium channel.<sup>[3]</sup> The abnormal myocardial sodium channels decrease sodium inflow currents, thus reducing the duration of normal action potentials. A number of conditions may induce Brugada pattern EKG such as fever, hyperkalemia, hypokalemia, hypercalcemia, cocaine abuse, and medications such as sodium channel blockers, heterocyclic antidepressants, and beta-adrenergic blockers.<sup>[4]</sup>

Hypokalemia may cause QT prolongation and other ventricular arrhythmias due to its effect of raising resting membrane potential and increases both the duration of the action potential and the refractory period leading to reentrant arrhythmias. It also increases the threshold potential as well as automaticity and decreases conductivity.<sup>[5,6]</sup> Although there is no definite value of serum potassium level that can induce Brugada pattern EKG, some case reports revealed hypokalemia may unmask Brugada pattern EKG.<sup>[7]</sup> In an animal model, it was revealed that loss of the action potential dome due to transient outward current (Ito)-mediated phase 1 of potassium in the right ventricle induces a transmural voltage gradient which accentuates ST-segment elevation which is similar to the mechanism of BS.<sup>[8]</sup> Therefore, hypokalemia may expand transmural dispersion of repolarization in the right ventricle leading to a Brugada pattern EKG.<sup>[9,10]</sup> EKG changes in hypokalemia are usually T wave inversion, appearance of U wave and ST-segment depression of 0.5 mm or more in lead II or leads V1, V2, and V3.<sup>[11]</sup>

The decision to follow-up with an electrophysiologist is determined by the patient's age, family history, prior episodes of



**Figure 1:** Coved ST-segment elevations with T wave inversions in leads V1 and V2



**Figure 2:** Disappearance of Brugada pattern electrocardiogram after normalization of potassium

syncope due to cardiac disorders, and patient's preferences. The consensus recommendations for the treatment of BS focus on implantable cardioverter-defibrillator (ICD) to abort ventricular arrhythmia. ICD was preferred over antiarrhythmic agents such as quinidine or amiodarone in patients with a history of SCD or syncope.<sup>[12]</sup>

## Conclusion

Hypokalemia may unmask or exacerbate Type 1 Brugada pattern EKG. Awareness of its appearance should be made by all physicians since patients with a family history of arrhythmia or SCD are at the high risk of developing SCD.

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## Conflicts of interest

There are no conflicts of interest.

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